# EXHIBIT B

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY
CAMDEN VICINAGE

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| IN RE: VALSARTAN, LOSARTAN, AND IRBESARTAN PRODUCTS LIABILITY LITIGATION | : | Honorable Renée Marie Bumb District Court Judge |
| THIS DOCUMENT RELATES TO Gaston Roberts, et al. v. Zhejiang Huahai Pharmaceutical Co., et al. | : | Case No. 1:20-cv-00946-RMB-SAK |

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MAY 8, 2025

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Remote Videotape Deposition, taken via Zoom, of GREGORY DIETTE, Ph.D., commencing at 11:03 a.m., on the above date, before Amanda Maslynsky-Miller, Certified Realtime Reporter and Court Reporter in and for the State of New Jersey.

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APPEARANCES:

NIGH GOLDENBERG RASO & VAUGHN, PLLC
BY: DANIEL NIGH, ESQUIRE
BY: STEPHANIE IKEN, LAW CLERK
1333 College Parkway
#1049
Gulf Breeze, Florida 32563
(850) 600-8090
dnigh@nighgoldenberg.com
siken@nighgoldenberg.com
Representing the Plaintiffs

KIRKLAND & ELLIS LLP
BY: JESSICA DAVIDSON, P.C., ESQUIRE
601 Lexington Avenue
New York, New York 10022
(212) 446-4800
jessica.davidson@kirkland.com
Representing the Defendant

GREENBERG TRAURIG LLP
BY: STEVEN M. HARKINS, ESQUIRE
Terminus 200
3333 Piedmont Road NE
Suite 2500
Atlanta, Georgia 30305
(678) 553-2100
harkinss@gtlaw.com
Representing the Defendants

ALSO PRESENT:
Bill Geigert, Videographer

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I N D E X

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E X H I B I T S

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NO. DESCRIPTION PAGE

No exhibits were marked.



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DEPOSITION SUPPORT INDEX

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Question Marked
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None



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(It is hereby stipulated and agreed by and among counsel that sealing, filing and certification are waived; and that all objections, except as to the form of the question, will be reserved until the time of trial.)

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VIDEO TECHNICIAN: Good morning. We are now on the record. My name is Bill Geigert. I'm a videographer for Golkow, a Veritext division. Today's date is May 8th, 2025. The time is 11:03 a.m.

This remote video deposition is being held in the matter of Valsartan, Losartan and Irbesartan Products Liability Litigation in the United States District Court for the District of New Jersey. The deponent is Dr. Gregory Diette.

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All parties to this deposition are appearing remotely and have agreed to the witness being sworn in remotely.

Due to the nature of remote reporting, please pause briefly before speaking even to ensure all parties are heard completely.

All counsel will be noted on the stenographic record. The court reporter is Amanda Miller, and she will now swear in the witness.

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GREGORY DIETTE, Ph.D., after having been duly sworn, was examined and testified as follows:

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EXAMINATION

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BY ATTORNEY NIGH:

Q. Good morning, Doctor. Can you please state your name and spell your last name, please?

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A. Sure. It's Gregory Diette, D-I-E-T-T-E.

Q. And, Doctor, you have -- you've been in many depositions, correct?

A. I have.

Q. Okay. I'll just go through a couple of the ground rules.

First off, it's not an endurance test. If you need a break at any time, just let me know and we'll take a break, okay?

A. Sure thing.

Q. And, second, if I ask a question and you don't understand the question, just ask me to repeat it or I can rephrase it for you.

But if you answer the question, I'm going to assume that you understood the question.

Is that fair?

A. It is fair.

Q. Okay. Doctor, when did you first start your work in relation to this case?

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ATTORNEY DAVIDSON: So, sorry, Daniel. I'm not going to object a lot today. I want to be efficient.

When you lean back, your voice becomes a little bit weak. And so it's, like, hard to hear you.

ATTORNEY NIGH: Okay.

ATTORNEY DAVIDSON: I don't know why. Something about your microphone. If you can just get, like, a little bit closer to your microphone.

And, hopefully, you won't hear for me for a long time after this.

ATTORNEY NIGH: Okay.

BY ATTORNEY NIGH:

Q. Doctor, when did you first start your work in this case?

A. It was last calendar year. And I think it was approximately November or so of last year.

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Q. Okay. And so November of 2024?

A. That's right.

Q. And approximately how many hours have you worked on this case?

A. I don't know if I'd have a good estimate. I can tell from -- like, I don't know, do you have any, like, invoices or anything?

Q. Without looking at the invoices, do you have any sort of estimate?

A. Not a good one. No, I think -- I mean, I think the invoices would be, you know, reasonably accurate at least for the -- you know, for the months that there's an invoice.

Q. Do you believe that you've spent more than a total of 100 hours in this case?

A. I don't think it's as much as that.

Q. Do you think you've spent somewhere between 50 to 100 hours?

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A. It would be in that range, yes.
Q. And how much do you bill per hour?
A. It's $600 per hour.
Q. Okay. And, Doctor, I've reviewed your report. And other than the case caption, I don't see Mr. Roberts' name anywhere in the report.
Does that sound accurate?
A. It does.
Q. Did you review Mr. Roberts' medical records?
A. Not -- not in detail. I received some, and I just -- I mostly just skimmed a couple of places, I think mostly, actually, in some of the other expert reports, just to get the general -- you know, my -- a general understanding of what the case was about.
But I did not scrutinize his medical records.
Q. Did you review any of his prescription records?

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A. I did not.
Q. Did you review any of the testimony of the treating doctors in this case?
A. No.
Q. Did you review any of the testimony of the prescribing doctors?
A. Isn't that the same thing?
Q. Treating, prescribing, sometimes people call it the same thing.
A. I see.
Well, either way. So neither one.
Q. Okay. And did you review the transcript of his -- the deposition transcript of his wife?
A. No.
Q. Okay. Did you give any opinions today that are specific to Mr. Roberts?
A. Not yet.
Q. Okay. Suffice it to say your opinions are related to general causation, correct?

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A. Oh, I think -- I'm sorry, I -- I just listened to the tense. I mean, you said did I, and I don't think I've given any opinions yet.
So I just -- that's all. I'm just trying to be literal.
I'd say there's one opinion that might be specifically about him. But otherwise, I would say they're all general.
Q. And what is the one opinion specific to him?
A. From looking at the materials, I saw that, you know, different experts estimated his latency from the time of use of the NDMA-contaminated valsartan to be about 1.86 years. And it seemed to me that the latency was shorter than anything I could find, you know, in the scientific literature.
Q. So other than latency, did you give any other opinions that are specific to Mr. Roberts' case?

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A. I don't plan to.
Q. Doctor, in evaluating epidemiological studies regarding toxic substances, it's important that you understand the characteristics of that toxic substance, correct?
A. To a certain extent, yes.
Q. What research did you undertake to try to understand NDMA?
A. Well, I looked at a variety of sources. I mean, I'm somewhat familiar with nitrosamines just from, you know, having done some other work.
But for NDMA in particular, it wasn't just one source. And I don't know if I remember them all.
But it certainly included, like, IARC monographs, you know, which usually do a nice job of describing what the -- the substance is. In, you know, each of the epidemiological studies there's usually a little bit of information about what NDMA is as well. I think there's an ATSDR document that

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provides information about that as well.
I don't know if I'll think on the spot of every single source. But, you know, there's an oncology textbook that I like and refer to. The Weinberg textbook. And they describe it, as well, in relationship to, you know, information about potential carcinogens and so forth.
There may be other sources as well.
Q. Did you review the general causation reports submitted in the valsartan case?
A. I guess can you be specific? Because I don't know how many there were.
Q. Other than -- other than Dr. Siddiqui's report, Dr. Zauer's report and Dr. Bruce's report did you review any other expert reports in this case?
A. So expert reports from Dr. Mamood, I think. And I think -- that seemed like it was more specific causation, perhaps, but I think maybe some general causation.

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And then the one other doctor. I don't know if I referred to it in my report. There's another -- another defense expert, the guy at Penn who is a biologist.
Q. Did you review the report of Dr. Panigraphy?
A. Can you say the name again? I'm sorry.
Q. Panigraphy.
A. I may have seen it back months ago, but I don't recall that.
Q. Did you review the report of Dr. Etmanon?
A. Again, I couldn't quite hear the name.
Q. Etmanon?
A. It doesn't sound familiar.
Q. Did you review the report of Dr. Gibbs?
A. If I did, I don't recall.
Q. Doctor, NDMA is a complete carcinogen, correct?
A. I don't know if that's true

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or not, particularly in the context of humans.
Q. What is a complete carcinogen?
A. You may get a different answer from somebody whose, you know, field is cancer biology.
But, to me, it's a carcinogen that can either promote, promote -- well, it can do both, it can initiate and then also promote the cancer.
Q. Have you reviewed materials that suggest that NDMA can promote cancers?
A. In humans. I mean, I looked at the ATSDR statement. And, you know, they looked at whether it was a genotoxic agent, for example, and pointed out that with the exception of one case report, that there's not human studies.
So, you know, I don't -- I don't know that -- otherwise that it is.
Q. Animal studies have shown

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that NDMA can act as a promoter in animals, correct?
A. That's my understanding. And that's without doing a deep dive into the -- you know, each and every animal study, but looking at other entities that summarize that, such as ATSDR.
Q. Do you have any reason to believe that the -- that if NDMA can act as a promoter in animals that it can't act as a promoter in humans?
A. I -- I can't say one way or the other whether that would be true.
Q. What does it mean for a carcinogen to act as a promoter?
A. Something that essentially can encourage the growth of an existing tumor.
Q. Doctor, have you reviewed key characteristics of carcinogens?
A. Ever in my life? I'm sure.
Q. Do you know what the ten key characteristics of carcinogens are? Does that sound familiar to you?

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A. I know there's a list that IARC uses, and I don't know if it's the same one. If it is the same list, then I've seen them at one time. But I can't recite them.
Q. Have you evaluated or considered any materials that look at the key characteristics of NDMA?
A. I have not.
Q. Doctor, in toxicology have you heard of the adage that dose makes the poison?
A. Of course.
Q. And what does that mean?
A. Well, that -- it means a couple of things. I think, you know, sometimes people use that to refer to something more along the lines of dose response.
But I think the more general meaning is that, you know, virtually any substance can be toxic at a sufficient dose, at some high dose.
And so it refers to the fact

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that -- you know, that a substance that can cause, you know, harm, for example, at a high dose doesn't necessarily cause it at a low dose.
Q. Doctor, do you know the amount of NDMA that was in valsartan pills?
A. No. I read different information about it from different expert reports. But I don't have an opinion about what it is.
Q. Can you describe the amount of NDMA that was in Mr. Roberts' pills?
A. No. I mean, I could certainly, you know, reread the portions of different expert reports that articulated that.
But I haven't tried to commit that to memory.
Q. Can you describe the amount of NDMA that are in valsartan pills in general?
A. I think that's what I was answering in the question before.

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But, again, I could -- I could read it again. It's in some of the documents I've looked at. But I have not made any effort to commit it to memory.
Q. Have you looked at the amount of NDMA that's in ranitidine?
A. No, same -- same issue. I've seen, you know, writings about it. But I've not tried to commit it to memory.
Q. Do you know the amount of NDMA that's in nizatidine?
A. I have no idea.
Q. Have you looked to compare the amount of NDMA that's in nizatidine versus the amount of NDMA that's in ranitidine?
A. No. I can't imagine what purpose it would serve me. But I certainly haven't done it.
Q. Have you looked to compare the amount of NDMA that's in nizatidine compared to the amount of NDMA that's in valsartan?

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A. I haven't tried to compare the amount of NDMA in any two different, you know, pills or products.
Q. My next question is, have you attempted to compare the amount of NDMA that's in ranitidine versus the amount of NDMA that's in valsartan?
A. No. I mean, same answer. I mean, I haven't -- haven't made any effort to try to compare, you know, different types of pills.
Q. Doctor, have you -- you mentioned in your report an unpublished study.
Do you recall that?
A. The Lee study?
Q. Yes.
A. Yes, I do.
Q. And did you only review the abstract for that study?
A. No, there's a -- there's a preprint that's available online. So there's both, like, an abstract and, like, a -- you know, it sort of has the

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format of a full-fledged manuscript and so forth.

But it's a -- it's, like, a preprint, I think they call it, that has not gone through peer review.

Q. Did you notice any differences between the preprint and the abstract?

A. Well, the abstract I saw was tiny. I mean, there's, like, so much more information in the -- in the, you know, manuscript version of it.

Q. Did you notice any differences in the preprint manuscript that conflicted with the information provided in the abstract?

A. I don't recall that.

Q. Did you -- did you look to compare those two sources to see if they had conflicting information?

A. Well, no, I didn't hold them side by side. I read them each and just tried to glean what information each one had.

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Q. When you gave your opinions, did you rely on the abstract or did you rely on the preprint?

A. So I think the preprint has, like, a -- sort of a more fulsome description of what the methods are and certainly, you know, have more results and they have a -- you know, a discussion section that's longer.

It's -- you know, I don't put a lot of stock in either because they're not peer reviewed. You know, they're just something that exists in the world.

So I didn't really compare the weight of one or the other. But the one that has more information certainly has more information.

Q. And when you say you don't put a lot of "stock in either," what do you mean by that?

A. Oh, because -- because these aren't part of the peer-review literature, right. So to me -- to me,

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really, the meat of what I would have available to me for understanding the epidemiologic evidence on a topic, you know, would come through the peer-review publication, you know, pipeline.

And so far these have not gone through that.

Q. Have you looked to -- or, sorry. Strike that.

Do you know whether or not the Lee paper was submitted in peer review?

A. There's no information about that.

Q. Okay.

A. At least not available to me.

But -- I'm sorry, I'm just -- I'm just trying to say, yeah, so I don't -- I don't have any idea, like, whether it's been submitted.

Q. Doctor, did you look or rely upon any abstracts for the ranitidine studies that you looked at?

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A. And I guess by "abstracts" -- I just -- I just want to make it clear, it's not a big deal, but almost all the peer-review papers have a section called the abstract, right. So the abstract is kind of baked into a lot of those.

I don't -- I don't recall that there were any that were just simply conference proceedings in that sort of abstract.

Q. Did you rely on any unpublished studies in regards to ranitidine?

A. I don't believe so.

Q. Do you know if there were any?

A. I'm not aware.

Q. Did you search for those?

A. Well, I searched for ranitidine studies, and nothing else popped up other than what I -- you know, then what I've provided, you know, in my report.

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Q. Doctor, did you evaluate potential bias of any of the studies that you cited?
A. Of course.
Q. And which studies that you cited to had potential conflicts of interest?
A. Oh, that kind of bias?
Q. Yes.
A. Because bias is an epidemiologic term, right. So when I answered your question yes, I was talking about biases meaning, like, selection bias, issues with confounding, you know, other -- other sorts of bias.
In terms of conflicts of interest, I read that in every paper. But I didn't -- I didn't store, like, a memory of, you know, which ones did or didn't report a conflict of interest.
Q. Do you know if any of the studies that you cited to reported conflicts of interest?
A. I'd have to look at each and

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every one. I just don't recall.
Q. Do you recall whether or not any of the studies were -- that you cited to were published by manufacturers of -- sorry. Strike that.
Doctor, when you look at studies, why is it important to evaluate the amount of follow-up that's included in studies where -- when they're looking at cancer?
A. So the "amount of follow-up" is a little bit vague. But I'll just tell you how I would -- I would tackle that and you can please just tell me if that's not what you were after.
Because the amount can mean the length of time, for example. It can also mean the proportion of people that entered the study that also provided data at follow-up time periods.
And so, you know, each of those is an important concept, right. If you start a study and, you know, 90 percent of the people aren't available

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for you to know what happened to them, you know, that's an issue.
The length of time, which for cancer studies, assuming that there's an exposure of interest and the outcome is cancer, then it would get into the issues of latency. And that would be important as well.
Q. In terms of cancer and the length of time, why would it be important to evaluate the amount of follow-up included in the study?
A. So for the length of time, you know, if it's known or suspected what the latency is between the timing of an exposure and the outcome, meaning, like, the incidence or the diagnosis of cancer, it would be important to have a sufficient amount of time go by in order to find that -- that relationship.
Q. In order to know the true effect or the true point estimate that a carcinogen would have on cancer, is it true that it would be important to have

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follow-up for decades?
A. Well, it could include decades. It wouldn't automatically have to include, you know, decades for every single sort of cancer.
You know, for example, you know, some of the cancers, like leukemia, on average have much shorter latencies.
And so, obviously, you could learn more information from a study that had decades of follow-up, but it wouldn't be necessary, you know, in order to learn some information.
Q. Other than blood cancers, would it be important to have decades of follow-up in order to assess the true point estimates for cancer-related carcinogens?
A. I think so. I mean, it's -- you know, so part of it is -- just one of the issues is, I think, for many cancers it's not known what the latency is.
And latency is also very specific, right. So the latency between

one exposure may not be the same latency between a different exposure and that same cancer.

So, you know, if there's knowledge about what that latency is in any of those conditions, you would at least want there to be enough time to go by for that -- for that latency to be reflected.

You know, decades would be better. I mean, decades is always better, right, in terms of just being able to have more information.

Q. Doctor, in terms of latency, the latency in a general population is a different -- different analysis than the latency than any individual person can have in terms of getting cancer, correct?

ATTORNEY DAVIDSON: I'm sorry. I'm going to object, because I didn't understand the question. Can you restate?

THE WITNESS: I was going to say, I didn't quite understand



either. I apologize.

BY ATTORNEY NIGH:

Q. Doctor, in understanding the latency in a population, that is a different analysis than the latency that an individual can have, correct?

ATTORNEY DAVIDSON: Still going to object to the question as vague.

THE WITNESS: Yeah, I'll take a crack at it. I mean, I guess, I mean, a population study if it's designed properly could give you information on what the average or range of latencies is that can occur in human beings, you know, between the exposure and the development or the recognition of that cancer.

For an individual, you could calculate it. But if you wanted to understand it, it would make sense to relate that to what the population-based evidence is for



latency.

BY ATTORNEY NIGH:

Q. And in a population of people, there can be a median time for latency, correct?

A. Exactly.

Q. And then there can be a one standard deviation out from that median, correct?

A. True.

Q. There can be two standard deviations out from that median, correct?

A. Well, there can be any distribution around that median. But, yeah.

But it includes, you know, math that you could do along that -- along those lines, too.

Q. So when you're discussing latency and cancer, there could be a whole distribution in terms of how long it takes for people to get cancer, correct?

A. Of course.



Q. And some people can get cancer much quicker than the median and some people can get it much longer than the median, correct?

A. I don't know. I mean, I would have to consider cancer by cancer and what the evidence is.

But certainly there can be a range that includes shorter periods and longer periods.

Q. For liver cancer, have you considered what the first standard deviation would be for latency --

A. I haven't --

Q. -- in a population?

A. So I haven't tried to do it for a standard deviation. I've tried to look and see, you know, what's been published, which is not much on latency.

First of all, I don't find anything on latency between NDMA-contaminated valsartan and liver cancer. So that I can't answer.

You know, there's -- there's

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information about vinyl chloride that looks like it's about 12 years would be the typical distribution.

And then, if you'll see, I cited a -- an article, or at least a statement from the CDC, you know, where they were trying to decide who could be compensated for illnesses that could have resulted from participating in the 9/11, you know, cleanup and so forth. And, you know, they -- they cited that 12 year.

They also cited an article where someone had tried to create a distribution of, like, basically, the ranges of cancers that are solid cancers.

And in particular, they came up with an estimate -- I think it was, like, about -- it was, like, 10.68 or something, it was -- approximately, like, 11 years in general from that.

And then I think overall they said that they cited to -- I think it was about, like, 6.8 to, like, 65 or some really long number for solid cancers

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more generally, you know, that wouldn't be specific to liver cancer.

So that was the kind of information I was able to find.

Q. For vinyl chloride, were you able to find what the one standard deviation would be in latency?

A. You know, I don't know if that's in the -- in the document. But I certainly didn't memorize it if it is.

Q. Did you look to see, for vinyl chloride, what two standard deviations was for latency in liver cancer?

A. I can't tell you anything about the distribution in terms of these questions in terms of standard deviations.

And, you know, to be frank, I don't even know if it's a bell-shaped curve, which would somehow fit into the standard deviation idea that we're discussing.

Q. Did you -- do you have any

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idea what the distribution curve looks like for liver cancer in terms of latency?

A. No, I don't. I looked to see what the -- what the reported latency was. And so, you know, what they reported was 12 years by citing the article in that World Trade Center document. And then for a different author, they cited the nearly 11 -- 11 year.

Q. And that 12 years and the 11 years, that's the average time in terms of latency for liver cancer, correct?

A. For liver.

But they gave a range. So not -- not a distribution around one median, but they gave a range of latencies for solid cancers in general. And I'd have to look back to get the exact number, but it was something like 6.8 all the way up to, like, 50 or 60 years.

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So that was --

Q. Do you recall -- sorry, go ahead. I didn't realize you weren't done.

A. No. That's okay.

So that was the range of latencies that they were reporting.

Q. And that range of latencies, do you recall the percentiles that they were reporting?

A. I don't know what that means.

Q. Well, the top -- was it the 25 percent, 75 percentile, in that range that they reported?

A. I don't think that's the way that they reported it.

I think -- I think they were -- well, what they were after, right, was the range of plausible latencies. You know, they weren't -- this wasn't an exercise in plotting distributions, right.

The idea was, who's going to

get compensated for a tumor, right. And so they -- they used the information they could find for the few tumors where it was available to see what the range of plausible -- what the range of plausible latencies was.

And then they chose four for solid tumors. And I think, although they didn't articulate it, it seemed to be that was an effort to try to, you know, if anything, err on the low side so that people would get compensated from the World Trade Center fund.

Q. Right. And that range of plausible plausibility, do you know what that range covered? Do you think that it covered the entire distribution of latency that they looked at?

A. I think it likely would, from the way that they wrote that statement.

Q. Okay. So you don't believe that that range of distribution that they were looking at was looking at a



percentile or only a portion of cancers in the center of the distribution?

A. I'm not aware that that happened. And it wouldn't make sense, you know, given the exercise that they were -- that they were performing.

Because they were trying to make sure, you know, that they didn't fail to compensate people who had a plausible latency. Really, that's what the exercise is about.

Q. Did you compare the differences in the key characteristics of NDMA versus the key characteristics of the carcinogens that were involved in the World Trade Center?

A. I don't -- I don't know how to do that. I mean, how do you compare them?

Q. Did you compare the key characteristics between NDMA with animal studies and the key characteristics of the carcinogens in animal studies for the World Trade Center?



A. I wouldn't -- I wouldn't know where to begin to do what you're asking.

Q. Did you compare the aggressiveness of NDMA, in terms of initiating and promoting tumors, versus the aggressiveness of the carcinogens that were involved in the World Trade Center?

A. Everything we're talking about is about human beings. And I'm not aware of studies that document something that's so-called aggressiveness for NDMA and liver cancer.

Q. Did you compare the aggressiveness of NDMA in initiating and promoting tumors in animal studies versus the aggressiveness of the carcinogens that were involved in the World Trade Center in animal studies?

A. So I don't know if aggressiveness is a technical term.

But in any case, I mean, if we're talking about looking at



characteristics of animal studies, I think that belongs to a different type of scientist than myself.

Q. Okay. Did you review any of the reports that looked at those comparisons in this case?

A. In which comparisons?

Q. The comparisons between NDMA and the carcinogens that were involved in the World Trade Center?

A. I don't recall seeing anything along those lines.

Q. Do you have any expert reports or opinions that you can rely on for that sort of exercise?

A. I don't think so. I mean, if you pointed me to one and it's something that I've read, I'd be happy to reread it. But I don't -- I don't recall seeing that.

Q. Doctor, the time or latency can be different in humans depending on the type of carcinogen, correct?

A. Yes, of course.

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Q. Did you do anything to understand or look to see how the time may be different, in terms of latency, for NDMA versus other carcinogens?
A. There's nothing to look at. There are no studies of NDMA that show what the latency is with liver cancer. So there's literally nothing to examine.
Q. Did you look at animal studies in any way for that information?
A. No. And that's a different field. That's not -- I mean, you have not asked me yet what kinds of backgrounds I have and don't have.
But I think if you asked me if I'm a toxicologist, which maybe you'll get to, I'll tell you I'm not a toxicologist. I don't do animal research.
So there's a couple of -- a couple of things that point to a different type of expert that may have the capacity to do that. I'm not sure if they do. But that's not my capacity.

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Q. So in terms of evaluating NDMA versus the other carcinogens that you looked at in terms of latency, you wouldn't have any information to evaluate those differences, correct?
A. There's nothing to look at because there's no information about latency in humans and NDMA. So there's literally nothing to compare it with.
Q. You wouldn't be able to do that comparison in terms of looking at animal studies, correct?
A. I just -- I think I just explained that that's not my capacity.
And it's -- and -- you know, and, to be frank, you know, if we're talking about rats, you know, rats aren't humans. And so I don't know what that would teach me. Rats don't live 70 years. They live two years, right.
So timing issues in rats, you know, wouldn't really tell me in particular much about what the human experience is.

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Q. Do you have any idea in which animals they've measured latency for NDMA?
A. No. I've read a little bit of information that mostly comes from other treatises. But I haven't made any study of that.
Q. Doctor, you previously explained loss of follow-up.
What does that mean?
A. In a cohort study, that people who are enrolled at a particular point in time -- and we're talking about cohorts here, for the most part.
In a cohort study, it would be people that either can't be located or, for whatever reason, didn't participate in the follow-up in the study.
Q. In terms of loss of follow-up, what are some of the shortcomings when utilizing insurance claims databases for epidemiological studies?

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A. In terms of loss of follow-up or was that more general about the shortcomings?
Q. In terms of loss of follow-up.
A. Well, I think they would be generic, I mean, in the sense if there were people that you couldn't ascertain what their outcome was, you wouldn't have information about them, at least up until the point when they were lost to follow-up.
You know, you would certainly have information -- because there's a stream of time going by, right, from the initial moment that you create the cohort concept, and then people can contribute data for many years and then maybe not contribute at the end of the study.
So you -- you just wouldn't have information, you know, one way or the other, about their outcome.
Q. Doctor, in the United

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States, insurance that people have is oftentimes tied to their jobs, correct?
A. So there's -- employer-based insurance is one -- one format.
Q. And if -- in the U.S., if people switch jobs, they may switch insurances as well, correct?
A. That's my understanding.
Q. One of the shortcomings in utilizing claims -- insurance databases in the United States, may be that when people switch jobs, they would switch insurances and then you would have loss of follow-up in the studies, correct?
A. If that happens and there's no other way to sort of, like, you know, sort of bolt-on information from a separate insurance database, like, because it might be feasible.
But in general I agree.
Like, for whatever reason, whether it's switching jobs or, you know, that there's a different insurance company involved, if the data only resides with that

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particular source, then, sure, it's a shortcoming if you can't keep following people.
Q. And that also happens in other -- other -- not just the United States, but other countries as well, correct?
A. So I would only be guessing about that. I mean, I don't -- I'm not an, you know, expert in insurance policies and so forth in other countries.
Q. Do you know if that happens in Germany?
A. I have no idea. I think -- I mean, I don't -- I don't know for sure which -- you know, which countries have, for example, you know, uniform government insurance, which doesn't vary, you know, versus having private insurance.
So if it became important, I might be able to figure that out. But I certainly don't hold, you know, myself as an expert in insurance policies by country.

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Q. When you evaluated the studies that you looked at, did you evaluate that concept?
A. Whether they changed insurance companies?
Q. Right. Per country, the -- how often they change -- they change insurances and, therefore, you would have loss of follow-up due to that issue?
A. I didn't track in particular if that was the reason.
But just that I think in these studies, which were relatively short-term studies, I don't think the authors of the studies, for the most part, articulated that there was -- that that was a problem.
So I --
Q. Do you know --
A. I'm sorry.
I just didn't see that reported as a problem by the authors of the studies.
Q. Do you know which countries

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are known for having the least -- the least loss of follow-up due to change of insurances?
A. No.
Q. When evaluating cancer, that's an important issue to look at, in terms of loss of follow-up due to insurance, correct?
A. Any -- any outcome would be.
Q. But outcomes that have longer latency, that would be an important issue to look at, correct?
A. I'm not sure what that means. I'm sorry.
Q. The longer the latency, the more there is an issue -- the longer the latency of the outcome that you're evaluating, the more there's an issue in terms of loss of follow-up due to an insurance switch?
A. Yeah. Sure. It could be. I understand now. Thank you.
So, yeah, if you have an exposure and an outcome that you're

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tracking and there needs to be sufficient latency, and if loss to follow-up occurs, you know, within that latency time -- within the latency that you believe that you need in order to make the study have the information, then, sure, it would have an impact.

Q. And that sort of loss of follow-up due to switching of insurances, that would normally leave to Type II bias, correct?

A. If -- well, type -- it's not a bias. I mean, Type II refers to an error, right. It's not a bias.

And there's an error that can occur because you failed to demonstrate the -- the risk that might be there. But it's not a bias as much as it is an error.

Q. Loss of follow-up due to switching jobs and, therefore, switching insurances, that would lead toward bias toward the null, correct, more often than not?

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A. You know, in theory, it could. But the truth of it is you wouldn't know. You know, it could work either way.

Q. Have you looked at studies that evaluated whether or not loss of follow-up due to insurance issues more likely leads to bias toward the null versus false positive biases?

A. I don't -- I don't recall ever seeing a study that made that their -- you know, their topic.

But I may have seen something, you know, years ago.

Q. Have you reviewed any position papers that discuss that issue?

A. Not in recent times. I don't know if I have in the past. But, you know, definitely not recently.

Q. Do you have any reason to believe that loss of follow-up due to insurance switching would not more often lead to bias toward the null versus false positive bias?

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A. Well, so false positives aren't a bias, right, they're an error, right. So you wouldn't expect to get a false positive from loss to follow-up, right. That wouldn't be the reason.

The positive signal you got would occur prior to the person leaving the cohort. So if it's a false positive, that's a completely different issue and, I don't think, has anything to do with switching, you know, per se.

Unless, you know, you're switching in and out of one group that has, you know, better and worse quality data. But otherwise, I don't -- I don't think that's -- that's not something you really compare.

Q. Non-differential misclassification generally leads toward bias toward the null, correct?

A. That's the general belief, yes. But it's not a guarantee. It's a supposition.

Q. And one type of

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non-differential misclassification could be loss of follow-up due to switching insurances; is that correct?

A. Well, I don't know. Only in the sense that it could be. You know, but the -- but, you know, when you're talking about, you know, things that bias towards the null and that have non-differential, then it makes an assumption that the reason for switching is not informative, right. And so you would need that information, too.

If there's an informative reason for switching, then that might work the other way.

Q. Loss of insurance due to switching jobs generally leads to non-differential misclassification that leads towards bias toward the null, correct?

A. No. And I -- if some of these questions are meant to be different, I'm not sure I'm hearing the exact difference in them.

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I think I've kind of covered it in terms of the best answers I can give to these topics so far.

But if I'm missing what's different about that last question, I'm happy to take another crack at it.

Q. The difference between the last question versus the question I asked before is the reason for switching insurances.

So people are switching jobs, that would tend to lead to non-differential misclassification that would lead toward bias --

A. No, I wouldn't agree with that. I mean, you need to know more. I mean, why are they switching jobs? You know, there might be a lot of information on why they're switching jobs.

Maybe they're sick. Maybe they're sick and they can't keep working in the same job. Maybe they were fired because they come to work drunk, you know, and they get moved another place.

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So when you switch jobs, it's not a random event, usually, right. There's a reason for it. And so that information could have a big impact on, you know, what happens to the study.

Q. You just talked about a lot of maybes and this could happen.

But I was asking -- my question was generally, as you look at the range of ways that people switch jobs in a whole, in a population, doesn't that generally lead to non-differential misclassification that leads towards bias towards the null?

A. Yeah, I can't endorse that. I don't -- I don't think I have enough information to endorse that.

Q. Have you reviewed any studies that -- that look at this issue as to whether or not switching jobs and, therefore, switching insurances would tend to lead toward bias toward the null?

A. No. I think you asked that also. And I still don't -- I mean, I

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haven't -- I haven't done that. I mean, I'm not aware.

I may have seen something years ago. But I'm not aware of anything recently that I have seen.

Q. So you would not have considered that in your opinions that you gave here today, correct?

ATTORNEY DAVIDSON: I'm going to object. Like, I feel like you're just asking and answering the same questions over and over.

ATTORNEY NIGH: They're not the same questions. That's a different question.

BY ATTORNEY NIGH:

Q. Doctor, you can answer.

A. Sure.

ATTORNEY NIGH: And that's an inappropriate objection, by the way.

BY ATTORNEY NIGH:

Q. You can answer, Doctor.

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A. Sure. Thank you.

So, I mean, what it links to is that -- what I said earlier that I didn't see that the authors of the studies, you know, detected that as a particular concern in their studies.

You know, I looked for what -- you know, including what the authors of the studies found to be concerns in the studies that we're talking about.

So it's not that I didn't consider it. I just didn't see that it was an issue that was raised as important enough to change these studies.

Q. Do you have any way of knowing, in these various studies, which studies that would have affected more, that people would have switched insurances and, therefore, had loss of follow-up?

ATTORNEY DAVIDSON: I'm going to object. The question lacks foundation.

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THE WITNESS: If it's written in there, I have a way to find out, which is for us to look at the studies again.

But I don't recall, you know, something off the top of my head that would let me answer that question.

BY ATTORNEY NIGH:

Q. But if you're looking at each individual study versus a systematic review of the studies, how would you do the comparison?

A. Just trying to answer your question. I mean, I wouldn't have planned to do it in any case. I mean, not to rank order that way.

I'm literally just responding to a question that doesn't really make a lot of sense to me. But I'm just -- I'm just thinking -- since you asked it, I'm just trying to answer it.

Q. In your systematic review,

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did you evaluate which studies would have been more likely to have misclassification bias due to switching of insurances versus which studies would have been less likely to have that bias?

A. Yeah, I mean, my answer is going to be the same over and over again here.

Like, I didn't track that they saw that as a problem. I have the skill set to go back and look at the specific question, if you believe that there's something there for us to look at that would be fruitful.

But I certainly didn't memorize anything about these few studies that would tell me how to rank order them.

Q. What other non-differential misclassification biases did you consider?

A. I'd have to look back and look at each article to think -- to think that through.

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I mean, as I looked through each article, I looked for sort of strength and weaknesses. I don't know if I saw a lot of others that would be non-differential.

I know that the authors sometimes talk about things that they're hopeful for, you know, which is that they're hoping that confounders might be distributed similarly between groups.

But when I considered that, I actually saw evidence to the contrary, right. So when they would talk about, you know, important confounders, they often were not distributed the same between, like, for example, the contaminated user group and the non-contaminated user group. So I saw suppositions about things like that.

But it didn't seem like it held up when you actually look at the findings.

Q. Doctor, if users of both contaminated valsartan and uncontaminated

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valsartan were -- were included in both the test and control groups, that would also lead toward bias toward the null, correct?

A. So let me just correct you.

I know Dr. Sawyer referred to things as a test group. There were no test groups in these studies, right. A test group is a group that's experimented upon. And none of these were experiments.

But if you're talking about -- and then can you say the rest of it again? Because I'm sort of stuck on the -- like, the kind of misuse of the terminology.

Q. No problem.

Doctor, if studies evaluated users that were both exposed and unexposed and put them into both groups in the -- in the analysis, that would lead toward bias toward the null, correct?

A. And to clarify, so you're

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talking about, like, the same individual was in both categories at different time periods?
Q. No. The same individuals in both categories, period. Not at different time periods.
A. Oh, well, how could that work? I mean, you can only take one -- one medicine at a time, right, of the same type?
So you're saying people were, like, doubling up and taking uncontaminated and contaminated valsartan in the same time period?
A. Doctor, if a person took exposed medication one month and the next month they took unexposed medication, that person would be -- was included in both groups in the Gomm study and Mansouri study, correct?
A. That's what I was saying. That's how I was answering before when you asked it.
Q. Right. And that -- that

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leading of including that same person in both groups could -- could lead toward bias toward the null, correct?
A. It could.
Q. Did you look at any analyses where they excluded them from both groups?
A. I think -- I'll have to look at each study to remember. I think there was at least a study -- one study that did, like, a sensitivity analysis and looked at people who were in different of the groups of the type that you're talking about.
But I think that the -- I think that the overarching analyses for these studies was basically people divided into users versus non-users.
And I'm talking specifically about NDMA-contaminated valsartan.
Q. Doctor, have you reviewed any studies that show how conflict of interest of authors can affect results?
A. So studies that examine that

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phenomenon and show how it can -- I don't think I've seen a study.
I mean, I'm generally familiar with the concept and my own understanding and my colleagues' understandings generally about the topic.
I don't know if I've read an article that -- that's about that.
Q. Have you -- in forming your opinions in this case, did you consider any studies that looked at systematic reviews of papers published by authors who had conflict of interest versus papers that were published by authors who did not have conflict of interest?
A. So I don't -- you know, I don't recall, one by one, which, if any, had reported, you know, potential or actual conflicts of interest.
I read that at the time that I looked at each study. But I can't -- you know, I can't recall enough, you know, sitting here without looking at them to answer your exact question.

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Q. Doctor, what is the hierarchy of evidence?
A. Are we talking about epidemiological evidence?
Q. Yes.
A. So in general terms, it refers to different study designs that have, you know, more prominence than others.
Q. And what would be at the top of the hierarchy of epidemiological evidence?
A. So for primary studies, clinical trials would. In some schemes, then, there are also, you know, like, pooling-type studies, so meta-analyses, for example, can get ranked higher than clinical trials if they're pooling -- pooling those studies.
Q. After clinical trials and meta-analyses, what would be the next highest level in the hierarchy?
A. So then you get into observational epidemiologic studies and

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then cohort studies and then case-controlled studies.
Q. So for observational studies, that would include cohort and case-controlled studies, correct?
A. That's correct.
Q. And cohort studies would be higher in the hierarchy of evidence than case-controlled studies?
A. For some people, including myself. But they -- they occupy, like, a similar band, if you will.
You know, they're sort of, like, clustered together within -- you know, within the hierarchy.
Q. Why would you include cohort studies as higher than case-controlled studies in the hierarchy of evidence?
A. They have advantages. And, you know, the advantages that are important to me are, you know, ascertaining the exposure status prior to the development of the outcome.
And so they -- they're able

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to eliminate one very important bias, which is recall bias, which case-controlled studies are susceptible to.
Q. And recall bias can lead towards --
(Reporter clarification.)
BY ATTORNEY NIGH:
Q. -- a Type I error?
A. Yeah. So finding a signal that is reported that isn't -- isn't the truth. That's right.
Q. Doctor, you're not aware of any clinical trials that looked and assessed NDMA and valsartan, correct?
A. I have not seen any.
Q. Without the clinical trials, the next highest level of evidence in the hierarchy of evidence would be cohort observational studies, correct?
A. That's right.
Q. And there were multiple cohort studies conducted of looking at NDMA and valsartan, correct?

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A. There were three, if we just count the peer-reviewed -- the peer-reviewed publications.
Q. And the Pottegård study didn't have any liver cancer cases in the exposed or unexposed valsartan groups, correct?
A. That is correct.
Q. So you don't believe that was sufficiently powered to assess whether or not NDMA caused liver cancer, right?
A. It's not just an issue of power. You know, they had a short -- a short follow-on, you know, period.
I mean, it's -- you know, when people -- I think people misuse the term not having enough power to find something. Because, certainly, small studies can find things. And it's not impossible to find liver cancer even in a smaller study.
But, you know, the smaller size and short duration of follow-up

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would make it more challenging in order to -- you know, to be able to confidently exclude a risk from anything they were looking at.
Q. Any time a study has zero outcomes for both the exposed and unexposed, that would call into question the sufficiency of the power of the study, correct?
A. No, it doesn't have to. It may well be that there just aren't any of that outcome in the population that's being studied.
So it's not a guarantee that it's -- that it's not sufficiently powered.
Q. There's no way to perform a power calculation when there's zero subjects in both an exposed and unexposed for a particular outcome, correct?
A. It depends upon what you mean. I mean, there's some people that oversimplify the concept of a post hoc power calculation and what data you need.

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But you can certainly provide an estimate. I mean, if you had an estimate of what you thought the prevalence of the disease was in one group and what you expected it to be in the other group, you could provide an estimate of what a study of that size would be -- you know, what the power would be.

Q. In terms of trying to do that sort of power calculation, you have to come up with an estimate that's outside of the study, correct?

A. It would be -- right. It would be information that you -- that you already know or believe to be true.

And, again, you're talking about a post hoc calculation, coming in after the fact and doing the math.

Q. Doctor, it would be unethical to knowingly give people valsartan contaminated with NDMA in a clinical trial, correct?

A. I would say likely so.

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It depends, though, right. What's unethical -- because -- I mean, I -- I'm trying to anticipate, like, what the reason is for your question, right.

So if an investigator believed that the NDMA in the valsartan was sufficient to raise the risk for cancer and you were trying to study whether or not, you know, people developed cancer, that would clearly be unethical.

Q. Doctor, the valsartan epidemiological studies that you've reviewed, they measured the outcome in terms of diagnosis of liver cancer, correct?

A. That's right.

Q. Okay.

A. Well, I mean, diagnosis might be generous. It's the reporting in administrative database.

I mean, to me -- but I get your point. I am not trying to quibble. But I just mean, you know, a diagnosis,

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to me, comes from clinicians, for example.

And these were not clinician diagnoses, per se. These were -- these were based on codes that were reported in an administrative database.

Q. In order to report an ICD9 rescue type code, there would need to be some basis that liver cancer was detected, correct?

A. Hopefully. I mean, although, you know, it's not a guarantee, right.

And the reason I just say that is, is that, you know -- and I don't know who -- you know, who produced the information in these studies to choose the ICD9 or ICD10 codes.

But, you know, sometimes it could be as simple as, you know, a trained coder looking for the words "liver cancer." And, you know, I've seen examples, even in my own clinic, where you put, like, rule-out, you know,

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something and somebody picks up the thing you're trying to rule out as the -- as the thing, right.

So it's just not a guarantee. But you hope that, on average, that it does reflect, you know, some truth about somebody -- somebody somewhere in the clinical environment having made that diagnosis.

Q. What you just described, the -- somebody picking up rule-out, you know, to that degree, that's a sort of bias or error that would generally lead toward non-differential misclassification bias, correct?

A. I don't know. I mean, it depends. But it -- but it certainly could.

Q. Do you have any reason to believe that that sort of error would occur more often in a group of exposed individuals compared to unexposed in a cohort study?

A. Well, I don't know how many

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different programs there are, for example. Like, you know, some of these are national databases. And so errors, if there are errors, you know, could be confined to a particular, you know, center or particular region.

And so, you know, you could get quite a distortion from errors that aren't necessarily, you know, system-wide, you know, meaning across, you know, the entire country, like the whole country of Germany, for example.

So, you know, I mean, if there's errors that have something to do with who is doing the coding or where the -- the patients are that are receiving the codes, you know, there could be, you know, differential or non-differential bias.

Q. Do you have any reason to believe that that sort of error would have occurred more often with the exposed group compared to the unexposed group?

A. I mean, not based on these studies. There's no information provided within the studies that could -- could inform that.

Q. The NDMA epidemiological studies also measured the diagnosis of liver cancer, or attempted to, correct?

A. Correct. They attempted to.

Q. And the NDMA occupational study of Hidajat measured, as the outcome, death due to liver cancer, or attempted to, correct?

A. That's correct.

Q. Okay. In none of those valsartan or NDMA epidemiological studies did they attempt to assess whether undiagnosed patients actually had cancer but just were not diagnosed yet, correct?

A. So I think I understand.

I mean, at a -- at a high level they didn't do any validation, right. They didn't do any further inquiry into any medical record or any other source, right. They had no other information about the accuracy or the validity of the diagnoses that they thought they were recording.

But, I guess, you know, what you're suggesting is a different study, which none of these authors articulated, which would be -- in order to find what you're talking about, you know, you'd have to, for example, you know, do a scan or, you know, do whatever diagnostic testing is required to detect not-yet-diagnosed cancer.

So none of those described a methodology like that. They are passive recipients of whatever the coders, you know, put into the administrative active database.

Q. Right. There are other diseases and outcomes where they attempt to do that sort of study, where they -- even on asymptomatic patients that may have some sort of outcome that they're assessing, by drawing blood or doing scans, correct?

A. Oh, yeah. There's all sorts of studies where you can -- you can look for not-yet-clinically reported, you know, diagnoses. There's screening tests, for example.

Q. For example, when assessing what percentage of the population may have celiac disease, they drew blood of all patients, even those who were asymptomatic, to assess what percentage of the population has celiac disease, correct?

A. I believe you. I don't know the study you're referring to. But I believe you.

Q. Okay. But that didn't occur here with NDMA and valsartan for the NDMA epidemiological studies, correct?

A. There was no -- there was no attempt, in any of these studies, to link any person-level clinical information to -- you know, to the study database.

Q. Doctor, when you were assessing the ranitidine studies, many of those studies had active comparator

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designs, correct?
A. They did.
Q. Do you pronounce that as comparator or comparator?
A. I would say comparator. But I'm not sure I'm right. But that's -- that's the way I would say it.
Q. Okay. In looking at the active comparator studies, it would be important -- or what -- sorry. Strike that.
What's the purpose of an active comparator in design?
A. It's -- it's a very important, you know, issue, because -- particularly for medications that treat symptoms, you know, for example.
And if those symptoms are indicative of a disease that's in the causal pathway, right, to the outcome that you're looking at, then one of the ways to try to account for the risk of developing a disease would be to look at people who are on a similar medicine,

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which would treat the same symptoms which would have something to do with the risk of the disease you're studying.
And I can be specific if it helps because -- or maybe. Or not. I'll just wait for the question.
Q. Well, when comparing an active comparator, it would be important that one of the -- that the active comparator would have a similar disease profile compared to the drug that you're testing, correct?
A. You would hope so.
And, again, you're not testing, it, right. You're not testing the drug.
Q. Measuring?
A. Sure. No, I'm sorry, I'm just -- I'm just listening to the questions, and I'm just trying to -- I'm just trying to make sure I'm answering what you're asking.
Q. I understand. I understand.
So in -- Doctor, most of the

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ranitidine studies that had an active comparator compared ranitidine with famotidine, correct?
A. They had -- yeah. Famotidine is one. And then some of them also had proton pump -- proton pump inhibitors as well.
Q. Did you assess whether or not the users of famotidine were a more diseased population than users of ranitidine?
A. Whether they were more diseased?
Q. Yes.
A. In what way?
Q. In many ways.
Did you assess that in any way?
A. I may have at the time I was reading them. But I think that the main issue is just that they -- you know, that they're indications -- I mean, the assumptions the authors made, on average, is that the indications for the drugs,

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you know, wouldn't differ, you know, between ranitidine and famotidine -- famotidine and also the proton pump inhibitors.
So, I mean, that was an assumption, I think, rather than a fact.
Q. Did you assess any of the studies to see that famotidine users were more likely to use alcohol than ranitidine users?
A. If I saw it at the time, I don't recall it today.
Q. Do you recall if the studies demonstrated that famotidine users were more likely to have serious liver diseases than ranitidine users?
A. I don't recall that. But I'm happy to look at the -- at the studies again.
Q. And those sorts of outcomes could affect the results of those studies, correct?
A. So if -- if one of the groups had more sort of already existent

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disease, it could affect the outcomes.
Q. Did you do that same assessment for PPI users versus ranitidine users?
A. Well, I didn't separate it. I just looked to see what the active comparators were in the studies and the rationale for it.
Q. Doctor, when doing a systematic review, it's important not to cherry-pick the results that favor one conclusion and ignore the results that go against that conclusion, correct?
A. Yes. I like that concept.
Q. Doctor, nearly all observational studies have limitations, correct?
A. Yeah. Every study -- every study has limitations.
Q. And in nearly every study, the study authors in observational studies nearly always point out limitations in their studies, correct?
A. Most of them do. And they

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post that -- they point out some, but not always all.
ATTORNEY NIGH: We've been going for about an hour. Do you want to take a break at this point?
THE WITNESS: That would be great. Just, like, five minutes or something just to refill the coffee.
VIDEO TECHNICIAN: Off the record, 12:11.
- - -
(Whereupon, a brief recess was taken.)
- - -
VIDEO TECHNICIAN: We are back on the record at 12:41 p.m.
BY ATTORNEY NIGH:
Q. Doctor, I want to talk about your past testimony, okay?
A. Sure thing.
Q. In the past four years, you have testified over 60 times either in

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deposition or at trial, correct?
A. I don't know the number. But, I mean, if you have some testimony list and that's the count you made, I don't disagree with you.
Q. Okay. Does that sound accurate or in the ballpark?
A. Yeah, I would have estimated a little lower than that. But, I mean, you know, not -- not tremendously lower.
Q. Okay. And in those -- in the last four years, have you ever testified on behalf of plaintiffs?
A. Yes.
Q. Okay. And in which of those cases did you testify on behalf of plaintiffs?
A. I'd have to look at the -- at the list, which I don't have in front of me.
But, like, there was, like, one -- one that I can think of was a woman who had indoor mold and dampness in her home that led to worsening of her

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lung disease.
And there was at least one other case. I don't know if I can recall which one it was.
But I think there's -- I think there's at least two in terms of testimony for the last four years.
Q. Do you have your expert report in front of you?
A. I do.
Q. But you don't have your prior testimony?
A. That's right. Yeah, I mean, I just -- I have, like, a printout, like, a hard copy of my report.
Q. No problem.
Okay. Do you recall what the second case was where you testified on behalf of plaintiffs?
A. Oh, no. I mean, I'm trying to say, like, I would need to look at the list, you know, to see.
Q. Approximately -- or, I'm sorry. Strike that.

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When did you start doing consulting work for litigation?
A. At least -- at least 20 years ago, I think, was, like, the first case.
Q. Okay. And what percentage of your work would you -- in terms of litigation consulting in the past ten years would you say was on behalf of plaintiffs versus on behalf of defendants?
A. It's hard to -- so in aggregate, it's hard -- the math is hard.
But by category, I'd say that, like, where it's medical malpractice cases, it's been about half and half. You know, there -- there -- for certain topics, like indoor dampness and mold, I'd say it's probably also 50/50, maybe with a slight -- maybe slightly more for plaintiffs.
For, you know, separate issues -- like you'll see talc-related cases on there, it's always for

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defendants.
So it depends upon the -- you know, the topic and what the specifics of the case are.
Q. So in terms of -- have you ever been an expert in medical device cases?
A. I don't think so. You mean, where there's like -- yeah, I don't -- I don't think so.
Q. Like a hip or bladder, implant or those sorts of things?
A. No, I don't -- I don't think I've come across anything like that.
Q. So you spoke about talc.
In terms of cases that involved manufacturers of substance, whether it be, you know, drug manufacturers or other substances, how often do you testify on behalf of plaintiffs versus on behalf of defendants?
A. If it's manufacturers, I'd say -- and you mentioned -- like, I can't

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think back before ten years ago, necessarily.
But for the last ten years, I can't think of a case where I've testified for the plaintiffs.
Q. Approximately how much money have you earned in the past four years for litigation consulting work?
A. So I'd say it kind of varies year to year. I'm just going to try to do it roughly.
It's not the same every year. But I would say in a typical year between 300 and 5 or $600,000.
Q. Which pharmaceutical manufacturers or manufacturers of substances have you testified on behalf of?
A. So I don't -- like, I don't know about pharmaceutical. Like, maybe none, with the exception of Johnson & Johnson. Although, like, it's not in the role of what their pharmaceutical are, right, because we're talking about talc

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in those cases.
So, I mean, if you took Johnson & Johnson, obviously they're -- they're a pharmaceutical company also. But I -- I haven't testified, you know, on my matter relating to a pharmaceutical.
And I don't think that there's anything -- anything on that list that you would have that's about a pharmaceutical, you know, issue.
Q. What about in terms of toxic substances, which companies have you testified on behalf of in terms of toxic substances?
A. I think -- I mean, the best way to do it is to kind of read through that list that you have, because that's -- that's a list of testimony.
So I won't do a complete job, you know, just from memory.
But, like, Colgate, Palmolive, for example, Avon. I think, like, a couple of -- did you say

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manufacturers? Is that what you said?
Q. Companies or manufacturers.
A. Okay. Yeah. There's been a couple of, like, talc suppliers that I've been, like, co-retained some certain of those cases, like IMI Fabi, Incorporated. And there was another one some years ago that went bankrupt.
But whatever -- whatever is on the list that we provided is accurate. So the ones I'm not saying out loud are, you know, still true if they're on that list.
Q. I understand. I only have a list for four years. So I'm just asking you just, in your history of testifying, which companies.
A. Yeah. Well, it's the ones that are on that list. And off the top of my head, I can't think of ones that aren't on that list.
Q. Okay. So in the -- prior to the last four years, you can't think of any companies or manufacturers that you

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testified on behalf of?
A. I can't. I mean, it doesn't mean there aren't any. But I'm not -- I can't think of any that wouldn't be on that list from the last four years.
Q. Okay. Doctor, what is a confounding factor?
A. As a term in epidemiology, it sometimes is -- and that's what we're talking about, right, epidemiology?
Q. Yeah. What is a confounding factor in epidemiology?
A. Thank you. No, I'm sorry. Because I know some people use words in English that, you know, mean something else --
Q. No problem.
A. -- from my field.
So, like, a confounder or a confounding factor, it's, in -- in some schemes is considered a bias. And it's a factor that distorts the relationship between the two -- the true exposure and the outcome that you're studying.

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Q. What is a potential confounding factor?
A. It's the same thing. Although potential would have to do with the -- like, the investigator or the scientist, you know, based on what their knowledge is or their belief.
So something that's a potential might be one that hasn't been shown but might be suspected to be a confounder.
Q. Nearly all observational studies have potential confounding factors that are not able to be addressed in the study results, correct?
A. That's right.
Q. And just because some potential confounding factors are not ruled out, this does not necessarily make the study findings unreliable, correct?
A. Not just because some. I mean, there's -- there's a hierarchy in some ways, you know, some are more important than others.

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Q. How would you describe that hierarchy of which are more important than others?
A. Oh. So, like, an example would be -- it may be in my report.
But if you look at -- you know, if you look back -- back years ago, a lot of people who drank coffee also smoked cigarettes, right. So if you did a study of coffee drinkers and looked at lung cancer incidence, you would almost certainly find that coffee drinkers have a higher incidence of lung cancer.
But, you know, we know pretty confidently that coffee doesn't cause lung cancer. And we know pretty confidently that tobacco smoke does. And so if your study didn't account for tobacco, that would be an example of an important confounder, you know, if you missed that, since that's the true cause of the outcome you're looking at.
Q. And, generally, a confounding factor would be -- in the

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example you gave, would be people who drank coffee would more often smoke than people who did not drink coffee, correct?
A. In that example, that's right.
Q. And, generally speaking, when looking -- when looking at confounding factors, that would -- generally would be what is needed, that the one group more often has exposure to something than the other group, correct?
A. In order for it to function that way, that's right. There would be more of that actual cause in one group than another.
Q. For any of the valsartan studies that you analyzed, did you find any confirmed confounding factors?
A. So "confirmed" meaning that they affirmatively looked at known or suspected confounders that did influence the results?
A. Yes.
Q. Well, they don't break it

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down that way, right. I mean, I'd say in general, right, that -- you know, for example, like, some of the analyses were adjusted for, say, you know, age and sex. And, you know, older age is clearly a risk factor for most cancers, you know, just given the accumulation of time.
And so the -- you know, age would be a potential confounder. And then if you've accounted for it, then you've done a good job of sort of eliminating age as the -- as an actual confounder in the study.
But the -- I didn't see, like, enough detail in the reports to say, you know, how much each and every one of the studied confounders, whether potential or not, influenced the results.
Q. Okay. So you did not find any confirmed confounding factors for any of the valsartan studies that you analyzed, correct?
A. Not -- not individually. I mean, to the extent that you see that,

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like, a hazard ratio, after adjustment, is a different value, then you know that the -- you know, when you compare it to the unadjusted result, the adjusted result, if it's different, will have accounted for those confounders.
But I didn't -- I didn't see an analysis where they isolated a particular confounder where we could say, yeah, that one mattered or that one didn't matter.
Q. For any of the NDMA dietary studies that you analyzed, did you find any confirmed confounding factors?
A. None specific in that way that I'm describing, you know, where they isolated a particular -- a particular item.
Q. For the Hidajat study, did you find any confirmed confounding factors?
A. I don't know if I saw any. I mean -- I mean, what I mostly noticed was the -- you know, the absence of

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consideration of some of the most important confounding factors.
But, you know, again, I didn't see, you know, like, the sort of granular detail you'd need to say, okay this -- this particular factor was clearly a confounder because it was studied, you know, in isolation or one by one.
Q. And when you mention the confounding factors in Hidajat, you mean the potential confounding factors in Hidajat?
A. What I was referring to? Is that what you're asking?
Q. Yes.
A. Oh, yeah. So what they said in the paper is that they -- you know, they couldn't account for lifestyle factors, for example.
So since it was a study looking at liver cancer, among other things. They had no information about alcohol use. They didn't have tobacco

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use. So, I mean, as examples, those are two important confounders.

And then, you know, even though the preamble talks about multiple carcinogens being in the environment of the rubber workers, they only measured a modest number of the exposures, right. So that the other -- the other ones are left hanging out there as potential confounders, because they didn't measure them and they didn't account for them.

Q. So for the alcohol and smoking, those would be potential confounding factors in the Hidajat study, correct?

A. So not the way that I said initially. I guess it's a matter of semantics. And I just want to kind of clarify it. And if I am not answering it, please let me know and I'll try to -- try to do, you know, better.

So when I was talking about potential before, you know, an example might -- there might be, like, in

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general, like, let's say somebody wondered if eating chocolate bars was a risk factor. They don't know it, but it might be and maybe they have some good reason for thinking about it. So it's a potential confounder. It doesn't mean it's going to be. That's how I meant that particular issue.

So alcohol use clearly is a confounding factor in a study of liver cancer, you know, if you believe that alcohol use is a risk factor for -- for liver cancer. The -- so that's how I'm using it. It's a actual confounding factor.

But it can't be -- it can't be studied in the Hidajat study because they didn't -- they didn't measure it.

Q. When you use the terminology that it is a con -- that alcohol is a confounding factor in the study, you mean that alcohol is a risk factor?

A. That's the way that one functions. That's exactly right.

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So alcohol is a risk factor. That risk factor -- because it can cause or be on the causal pathway to the outcome, which is liver cancer, and it wasn't accounted for, it's still a confounder. We just can't know what the effect of it is in that study because they didn't -- they didn't measure it.

Q. But there's nothing suggesting that -- that people exposed to -- in the fourth quartile of NDMA would have drank more alcohol than people exposed in the first quartile of the NDMA in the Hidajat study, correct?

A. We can't know -- we can't know one way or the other.

I mean, I would say that, you know, just in general that, you know, workers that have kind of the most or the worst exposures often have clustering of other unfavorable factors.

And so that might be, you know, true in that study. But we don't -- we don't have information to say

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one way or the other.

Q. Do you have any reason to believe that people who would work in the vulcanizing department would drink more alcohol than people who worked in other departments in the factory?

A. Yeah. And not as a fact, right. But in -- I've seen in multiple worker studies where the people who are in the worst environment -- and I would assume the vulcanizing environment is not very good, it must smell terrible and it seems like it's a -- generally speaking, from the way that the descriptions are in that series of papers, you know, it doesn't sound like the one people would sort of work their way up to. You know, it might be the -- kind of the most menial or the most -- you know, for lack of a better word, most disgusting, right.

So it's -- and then people that are in those jobs often are of the lowest socioeconomic status or have the least power in the -- in the -- you know,

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in the company. And some habits go along with that, you know, including smoking, drinking and so forth.
Q. So is your belief that the people who worked in the curing and vulcanizing department were in the nastiest part of the factory compared to the other parts of the factory?
A. I don't know how to rank order them. But it certainly -- it certainly sounds like one that would be an undesirable place to work.
Especially early on, like, back before they lowered the -- the levels of the different chemicals.
Q. Well, how would you -- did you try to do any sort of analysis comparing curing and vulcanizing compared to the other departments in the factory?
A. No, I can't do that.
Q. Do you know whether or not other departments in the factory where -- the workers were exposed to more of other carcinogens than in the curing --

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A. Than --
Q. -- and vulcanizing department?
A. Sorry. I didn't mean to cut you off.
No, I don't think they reported that.
Q. Do you know whether or not other departments in the rubber industry were exposed to more rubber dust and rubber fumes than the vulcanizing -- vulcanizing and curing department?
A. I don't recall seeing it broken down that way.
Q. Do you know whether or not other departments in the factory were exposed to more of the carcinogen in more than the curing and vulcanizing department?
A. I don't know that. I don't know if it's in the paper that we can look at.
But I don't recall that.
Q. Is it something that you

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looked for?
A. I wasn't trying to compare the different departments.
I mean, really, my answer to your question was just about what I know about, you know, occupations and who works in certain -- you know, certain types of occupation and some unfavorable factors that go along with it.
Q. Have you seen any sort of suggestion that -- that shows that, actually, other departments in the rubber industry were exposed to more carcinogens than the curing and vulcanizing department?
A. I don't know anything about that.
Q. Doctor, you mentioned that workers in the rubber industry were exposed to several carcinogens other than NDMA.
Do you recall this?
A. I do.
Q. Which of those carcinogens

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do you believe are a risk factor for liver cancer?
A. So I'd have to see the list again. It's in the preamble.
I don't know off the top of my head. Because I know they mentioned, I think, benzene and heavy metals and a few others. And I'm not aware that those are risk factors for liver cancer, per se.
But there may be others. I'm not sure.
Q. Do you believe that workers in the rubber industry have an overall increased risk of liver cancer compared to those who do not work in the rubber industry?
A. I'd say maybe. I mean, based on that -- that one study, you know, they do calculate an elevated risk.
So I'd say, you know, if that's all the information that we have, that it's -- that it's possible.
You know, there was a

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meta-analysis that preceded that that -- you know, that looked at cancer risk across rubber workers and multiple studies, and it didn't show an elevation.

So I guess if we somehow hierarchically decide this one study is more important than the ones captured in the meta-analysis, then maybe it's true.

Q. When you say the one study, you're talking about the Hidajat study that showed an increased risk of liver cancer when exposed to NDMA?

A. That's right. That's the one we're talking about.

Q. And when you talk about the meta-analysis, I believe you're referring to the Boniol study in 2017?

A. Yeah, '17 or '18. But, yeah, the Boniol study.

Q. Do you recall, did you ever do any sort of systematic review to see if there were other meta-analyses or other studies that showed that workers in the rubber industry did not have an

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increased risk of liver cancer?

A. I have not done any systematic review on rubber workers.

I think that, you know, my -- given my task here about NDMA, I think I understand that the Hidajat study that we're talking about is the one that, you know, measured and discussed NDMA.

Q. When you're considering whether or not exposure to all these other carcinogens that people would have in a rubber factory and whether or not that might be a risk factor for liver cancer, wouldn't you want to look at studies that look at overall exposure of workers in the rubber factory compared to people who didn't work in the rubber factory for liver cancer?

A. It could be helpful. But, you know, the authors are doing their job, you know, of trying to inform the reader.

I mean, you know, the purpose of an epidemiologic study is to

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communicate a lot of things. And in the introduction, the authors chose to inform the readers that there are many other carcinogens in the environment. And they also made it clear that they didn't measure those.

And then in their discussion, they also make clear that they didn't -- they didn't create any multi-pollutant models. So it's very hard to know whether they isolated the effect of anything that they're reporting, including NDMA or rubber dust or rubber fumes or the rest.

Q. And I believe when you say "they" in your prior answer, you're referring to the Hidajat study authors?

A. That's the study I believe we're talking about, yeah. I'll try to say otherwise, if I think we're talking about a different study.

Q. I'm asking about carcinogens in a rubber factory altogether.

There's not a -- there's not

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a demonstration that shows that overall exposure to working in the rubber factory leads to an increased risk of liver cancer, correct?

A. Not according to the meta-analysis that I saw.

And, you know, just -- and to remind you that I said no, I've not done a systematic review to find other studies.

Q. And if this exposure to carcinogens in a rubber factory were actually leading to an increased risk in liver cancer, wouldn't those sorts of meta-analyses be the studies that you would look at to see whether exposure to those carcinogens was a risk factor for liver cancer?

A. Sure. I mean, they would be helpful. But they would also, then, create the sense of a surprise that a single study did find it.

So why would one study find a risk when all the rest of them didn't?

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Q. Well -- sorry. Go ahead.
A. No, I was going to say -- that's it. I mean, that's a sufficient answer.
Q. Hidajat was actually looking at exposure to NDMA and whether or not varying exposures to NDMA led to an increased risk of liver cancer, correct?
A. So they included NDMA.
But my point is if you -- you know, it wasn't because they didn't find an elevated risk of liver cancer, right. I mean, regardless of what the substance was, if studies are looking at the industry and they don't find it, right, it's baked into it, whatever those exposures are.
This -- the Hidajat study is the one that made an attempt to relate estimates of NDMA and, in a handful of other exposures, you know, to the risk of multiple cancers, including liver cancer.
Q. The meta-analyses, they're -- they were -- like Boniol,

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they're looking at overall exposure to working inside of a rubber industry compared to those who don't work in the rubber industry, correct?
A. That's right.
Q. They did not analyze whether or not increased exposure to NDMA versus lesser exposure to NDMA in the rubber factory led to an increased risk of cancer, correct?
A. That is correct.
Q. Only the Hidajat study looks at increased risk -- increased exposure to NDMA versus decreased risk of exposure, and then the decreased exposure of NDMA leads to an increased risk of liver cancer, correct?
A. And I just -- just for me to answer it as correct, I would have to change "leads to" to "associated with."
But otherwise, I agree with your -- your question.
Q. Other than the qualitative description that you gave about the

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vulcanizing and curing department previously, do you have any information that would suggest that people exposed to higher amounts of NDMA in the Hidajat study were exposed to higher amounts of other carcinogens compared to those who were exposed to lower amounts of NDMA?
A. We can't know, which is an incredibly important flaw in the study and an incredibly important flaw if we're trying to reach the judgment that NDMA is the culprit, right.
Because if you look at the way they said they were going to do their analyses, they did separate models for rubber dust, for example, and for rubber fumes, as another example. And then one other nitrosamine.
And by doing each one at a time, they're doing single-pollutant models that don't take into account the correlation between each of those either substances or circumstances.
And that's why, you know, at

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the end, when they finish with, you know, we didn't do multi-pollutant models and they didn't even think that they had the right statistical techniques to do it, it leaves one wondering.
I mean, this could be as simple as my example with coffee and lung cancer, right. They found something that's associated with an elevated risk, but they haven't been able to isolate it from the other -- other substances that they looked at.
Q. How did Hidajat address whether or not there were issues related to multi-pollutant?
A. They mentioned that if -- at the end, they said that you might want to do it, or something, I'm paraphrasing because I don't have it in front of me.
But they brought up the issue that it might be done but that they -- they themselves, I think, didn't have the current statistical capacity to do that sort of modeling.

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Q. Are you aware of whether or not they did any sort of analyses related to multi pollutants?
A. I think they studied multiple pollutants. But I can't tell from the study that they actually accounted for multiple pollutants in any one particular model.
So, for example, you know, they looked at an elevated risk related to rubber fumes, but I don't -- I can't tell from the analysis that that accounts for what the NDMA was and vice versa.
Q. Doctor, there were two Hidajat studies, correct?
A. So there's -- there may be more, but there's two I'm aware of. One had to do with, I guess, application of the job exposure matrix. But one was sort of, like, the main results of the -- you know, of the cancer risk.
If those -- if those are the two you're thinking of, that's what I'm thinking of.

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Q. Did you look at the -- did you analyze the study related to the job exposure?
A. I did. And there's another one that, like, pre -- precedes that, that's also cited in Hidajat, which is really the -- it's really the source of the job exposure matrix.
So that study -- and I'd have to look, but I'm blanking on the first author, but it's not Hidajat for that study. But I did -- I did read the other Hidajat study, if it's the one that -- if we're talking about the same thing.
Q. Doctor, do you believe that the Hidajat studies did not do any analysis to determine multi-pollutant issues?
A. Well, I can't tell that they did. If you look at the -- if you look at their method section, there's a part -- and, again, I'm going to have to visualize it in my mind, because I don't

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have the paper in front of me -- but where they talk about the models, for example, for NDMA and, like, rubber dust and rubber fumes. And they use the word "or." They said they adjusted for things and it said each of those "or," so not "and."
So if they -- if they had said that they adjusted for those -- like, you know, one factor, another factor "and" the third factor, that could imply, you know, multi-pollutant models.
But they said "or," which told me it was individual models all by themselves.
Q. So you don't recall any multi-pollutant analyses performed by Hidajat?
A. Not of the types we're talking about.
I mean, if they included something else in their models that might have been, you know, interesting. But they certainly didn't include the -- you

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know, the carcinogens that they mention at the beginning that they didn't measure.
And yeah -- I mean, I'll leave it at that.
I mean, I saw that, you know, ATSDR when they evaluated the study, too, you know, also mentioned, you know, the absence of multi-pollutant models.
Q. And when you say "not of the type" that -- that we mentioned or were discussing, do you recall any multi-pollutant analyses or any analyses regarding whether or not multiple pollutants were having an affect on their outcomes?
A. I don't. But I'm just wondering -- I mean, if you're looking at something that shows one of the pollutants that we're not as interested in, maybe that's in one of the models. I just don't recall.
But I'm talking about the

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models that include the topics of interest, you know, which are rubber dust and rubber fumes and the different nitrosamines.

And I know they had a separate, like, category where they summed together -- they had, like, a sum score for nitrosamines more generally.

But that also is not accounting for multiple pollutants. It's just pooling -- you know, pooling a few of a certain category into one analysis.

Q. So you don't recall any analyses that looked at whether or not multiple pollutants -- multiple pollutants were having an affect on NDMA?

A. No, I don't. But, you know, again, I don't have the papers in front of me. And I wish I had a memory where I could memorize every detail of every paper.

But I've read it, and I just don't recall it.

Q. Your opinion states that

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there is no multi-pollutant analyses or any analyses that shows whether or not any other carcinogen had an effect on NDMA, correct?

A. Well, or whether it modified the effect of NDMA. I mean, that's in particular what we're talking about, whether it's NDMA in isolation, accounting for the other pollutants of interest, whether or not it alone is the explanation for the elevated risk that's been calculated.

And I'm just taking that from their -- their methods section, from their discussion where they talk about it. And then also, you know, ATSDR sort of reiterating that.

ATTORNEY DAVIDSON: Dr. Diette, do you need the article to talk about it? Because you are entitled to look at it. This is not a memory test.

THE WITNESS: I could do that. Or, you know, if Mr. Nigh

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just wants to show me what he's talking about, I mean, I can react to it. But I --

BY ATTORNEY NIGH:

Q. It's not a memory test. But I can ask these questions based on your opinions.

So we don't have to show -- I don't have to produce articles here in your deposition.

A. I can locate it, though.

I'm just saying -- and I'll just say this just to get it out there. Like, if it's an important issue and we need to solve it now, that's fine.

I mean, having said this and if we go to trial, I'll be sure to look at it and I'll answer your question more deliberately if I don't have a chance to do it today.

Q. But in your -- in forming your opinions, you don't recall looking at any sort of multi-pollutant analyses or analyses that looked at whether other

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carcinogens may have had an effect on the NDMA analyses, correct?

A. Yeah. And we're talking about in the Hidajat study that reports the risks of different cancers, right? That particular one?

Q. Yes.

A. Yeah. No, I don't -- I don't recall that.

Q. Okay. Doctor, hepatocellular tumors are capable of being promoted in numerous ways, correct?

A. I imagine that is possible.

We're talking about human beings here?

Q. In human beings, yes.

A. I imagine that is possible. But that's not really my field. So I don't -- you know, I don't know to the extent to which that's true.

Q. Do you know whether some carcinogens are capable of promoting hepatocellular tumors?

A. I don't.

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Q. And that's not something that you sought out to research, correct?
A. No. It wouldn't have informed my opinions for this case.
Q. Doctor, are you aware of a recent body of literature concerning -- regarding cancer that focuses on when and how subclinical tumors progress to clinical diagnoses?
A. Well, I don't know about -- what you mean by "recent" literature.
I mean, there's -- you know, one of the most fundamental, you know, discoveries in the, you know, timespan of my career has been, you know, looking at colon polyps, for example, which are a precursor lesion that progress, you know, over a decade or more to become a carcinoma.
So that's not recent, I mean, but that's -- that's one of the earliest that I'm aware of.
Q. Doctor, did you -- did you look at any or research any literature

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that discusses how hepatocellular carcinomas go from being subclinical tumors to progressing to clinical diagnoses?
A. No, no. That's not my expertise and not my field.
Q. Did you consider that literature when making your opinions in this case?
A. Well, I don't know what literature you're referring to.
But as a concept, you know, we're talking about epidemiologic literature that as an endpoint of an actually discovered tumor.
So, you know, that's what I was focused on, which is what epidemiologic studies look at, you know, which is something that you can measure.
I guess if there were -- if there were some different body of literature looking at what we could call subclinical, maybe pre-diagnosed, you know, that might be interesting.

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But it wasn't -- that topic is not part of the body of literature that was relevant for this -- you know, for this case.
Q. So in forming your opinions, you did not consider literature that looked at when and how subclinical hepatocellular carcinomas progressed to clinical diagnoses?
A. I didn't. And it wouldn't -- it wouldn't help me.
Q. Doctor, carcinogens that have the ability to promote can also promote liver tumors, correct?
A. I don't know what that means. I mean, you're using "promote" twice in the same question.
Q. Yes.
Carcinogens that have promoter capabilities can also promote liver tumors, correct?
A. Well, I don't know if that's a generalization. I mean, it -- I mean, it's such a broad question.

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I mean, if you're saying, for example, that something that's capable of promoting growth of a skin cancer could automatically do that for a liver cancer, I certainly don't know that to be true.
Q. My question is focusing on liver tumors, not skin tumors.
So my question is, Doctor, are you aware of whether or not carcinogens that have promoter capabilities can also promote liver tumors in humans?
A. Yeah, but my answer is exactly correct for the way you're asking the question.
Because a carcinogen is something that's capable of causing a cancer in some part of the body under certain circumstances, which doesn't mean that it can do that in any part of the body under any circumstances.
So if you're asking a general question about a promoter, and

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let's say we agree that it's a promoter, because it's been found in some other context, you know, that wouldn't -- that wouldn't relate automatically to liver -- liver tumors.
Q. Doctor, are you aware that carcinogen -- are you aware of whether or not carcinogens can promote liver tumors in humans?
A. I think I answered that before. I don't have that knowledge.
Q. And are you aware of whether or not carcinogens can promote hepatocellular tumors in humans?
A. Yeah, same -- same answer.
ATTORNEY DAVIDSON: Objection. These questions are overbroad.
ATTORNEY NIGH: Jessica, please object to form.
BY ATTORNEY NIGH:
Q. Doctor, I'm sorry. I didn't get your answer.
ATTORNEY DAVIDSON: This is,

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like, literally exactly how you guys object. But we don't need to argue. You and I get along.
Let's move on.
ATTORNEY NIGH: Okay.
THE WITNESS: I said it was the same answer to -- like, to the prior question.
BY ATTORNEY NIGH:
Q. Got it.
Doctor, there were multiple dose response analyses -- sorry. Strike that.
Doctor, there were dose response analyses in the Gomm study and the Mansouri study, correct?
A. That's correct.
Q. In terms of milligram dosage per manufacturer of valsartan with NDMA or NDEA, do you have any idea what the market share was per manufacturer per dosage of milligram of valsartan with NDMA or NDEA was?
A. How would I know that? The

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answer is no. The short answer is no.
Q. Doctor, who is ZHP in this case?
A. My understanding is that they are a pharmaceutical manufacturer.
Q. And ZHP, that's Zhejiang Huahai Pharmaceuticals?
A. Yes.
Q. What is ZHP's role in this litigation?
A. I assume they're a defendant.
Q. Do you know why ZHP is involved in this litigation?
A. Not factually, no.
Q. Did you review any of plaintiff's complaints in this litigation?
A. The complaints?
Q. Yes.
A. I don't recall doing that. I don't know if I received a complaint, but I don't recall reviewing it.
Q. Doctor, what is an active

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pharmaceutical ingredient?
A. I'll give you my answer, which might not be the same as, you know, a pharmacologist or, you know, a Ph.D., you know, with a pharmacy background.
But, to me, it's the component, you know, within a dosage of a medication that has the activity that you're -- you know, you're looking to use for the effect.
Q. Doctor, do you know whether ZHP is an active pharmaceutical ingredient manufacturer?
A. I do not know that.
Q. What is a finished-dose manufacturer?
A. I don't know that terminology.
Q. Doctor, do you know which finished-dose manufacturers manufacture their valsartan using active pharmaceutical ingredients manufactured by ZHP?
A. I can't answer it because I

don't know the -- I don't know what that first term in that question means.
Q. Doctor, have you ever heard that finished-dose manufacturers utilize active pharmaceutical ingredients in manufacturing the pill?
A. I suppose if I knew what that term meant I might have an answer. But since I don't know what the term is, I don't see how I could answer a question like that.
Q. Do you know which manufacturers utilized ZHP's active pharmaceutical ingredients in their pills?
A. I have no idea.
ATTORNEY DAVIDSON:
Objection. Asked and answered.
BY ATTORNEY NIGH:
Q. Doctor, did you compare the levels of NDMA and NDEA in the valsartan of those manufacturers who used active pharmaceutical ingredients manufactured by ZHP with the levels of NDMA or NDEA of



those manufacturers who did not use active pharmaceutical ingredients manufactured by ZHP?
A. And the first verb was what? Did I analyze that?
Q. Yes.
A. I don't have that capacity. I mean, if there was a report you wanted to show me, I could maybe read it. But I don't have the capacity to do an analysis of that.
Q. Did you compare those levels in any way?
A. No. I'm not even sure what we're talking about.
Q. Do you have any idea of how the levels of NDMA and NDEA in the valsartan manufactured using the active pharmaceutical ingredients manufactured by ZHP compare with the levels of NDMA and NDEA of manufacturers of other valsartan pills?
A. I don't have any capacity to answer that question.



And I'm not asking you to reword it, because I don't know what you're talking about.
Q. Doctor, do you have any idea of which manufacturers of valsartan had higher levels of NDMA and NDEA in their valsartan?
A. I have -- I have no awareness one way or the other.
Q. Doctor, do you have any idea as to the magnitude of how much higher the levels of NDMA and NDEA were for some manufacturers of valsartan compared to other manufacturers of valsartan?
A. I don't know whether there were differences and some were higher. But I don't -- I don't have any basis to answer that question.
Q. Would you have any basis to answer whether or not some manufacturers had hundreds of times higher amounts of NDMA and NDEA in their products compared to other manufacturers of valsartan?
A. Not sitting here now based



on what I know.
Q. Doctor, do you know whether manufacturers of valsartan that contain much higher levels of NDMA and NDEA had a higher market share of lower valsartan dosages compared to the manufacturers of valsartan that contained much lower levels of NDMA and NDEA?
A. Absolutely not. I don't even know how to begin to answer a question like that.
Q. Wouldn't -- wouldn't that information be important in assessing dose response sought out by Gomm and Mansouri when they looked at milligrams of valsartan use in assessing dose response?
A. Not a bit. I mean, it's -- I'm -- I'm left with the publication that they have presented through peer review, and I can analyze the results of that publication.
But if there's other differences you're talking about, I think

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that's for somebody else to ask and potentially answer.
But I -- you know, the paper as it is, is complete, at least for the sake of me being able to analyze it.
Q. But, Doctor, if -- if the manufacturers who had much higher levels of NDMA and NDEA, had a much higher market share of 40 milligrams, for example, than the manufacturers of -- that had lower levels, much lower levels of NDMA and NDEA, wouldn't that affect the dose response analysis?
ATTORNEY DAVIDSON: Objection. Improper hypothetical.
THE WITNESS: I mean, there's so much missing from that for me. Like, I don't even know what you're implying. Like, whether you're implying that there are some -- some of the pills in this study -- or that the study didn't capture certain pills from other manufacturers.

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I don't know if that's what you're saying. I don't know if you're saying within the study there were different manufacturers.
I have no idea. I mean, literally, as an epidemiologist, the only thing I can do is look at the results as they're presented by the investigators that have been through the peer-review process and interpret what they've written.
BY ATTORNEY NIGH:
Q. But you're doing a systematic review.
So you can actually compare -- you can look at other literature and other sources of information when you compare results within a single study, correct?
A. No. I mean, a systematic review, as you describe it, is a review of the actual other epidemiologic study.

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A systematic review doesn't include looking at all of the different scientific issues, you know -- and, particularly, I don't even know if these are scientific issues. These sound like product issues or something to do with, you know, somebody who knows something about different manufacturing processes or who knows what.
But it's -- all of these things pertain to something that's not in my field.
Q. But dose response analyses that were performed in Gomm and Mansouri, they're looking at the amount dose of valsartan not the amount of NDMA that people ingested in those studies, correct?
A. So they're starting with that. But they made some assumptions about what they believed that the NDMA dose would be.
But they're -- but they literally have, because this is -- I

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mean, for better or worse, this is what you're left with in a pharmacoepidemiology study, right, is you have dispensations of a product, and that's their exposure information. Their dispensations. So whatever is in those dispensation is baked into it, right.
I didn't see that either study did an analysis of individual pills to see what's in it or not. They are, you know, it's -- whether it's a weakness or not, it's something that isn't available in a pharmacoepidemiology study where you just literally look at pharmacy dispensations.
Q. Doctor, the Gomm and Mansouri studies also looked at whether or not using valsartan for three or more years led to an increased risk comparing exposure to unexposed, correct?
A. So Gomm three -- I mean, you're combining the two, right, so it's Gomm three and the Mansouri, you know, more than three.

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So it's correct, if you -- if you phrase it that way.
Q. Okay. And when they looked at three or more than three they found a higher increased risk compared to overall -- their overall analyses, correct?
A. So in one of the studies there was a numerically higher hazard that was no longer statistically significant. So I think one could appropriately argue they found no risk with the longer duration.
Q. Okay. You believe that just because it's not statistically significant that that can be argued that no risk was found?
A. Yeah. I think -- you know, the thing is with a study is that, you know, hopefully, they're not just providing statistical significance for no reason whatsoever, right.
I mean, the information about the 95 percent confidence intervals

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and the P value is there, right. It's required by most journals, and most people know how to interpret it.
So conventionally if you have a risk that's not statistically significant, you don't automatically say you've identified a risk factor for something. You would say I found a non-significant elevation or a non-significant decrease.
But that's not the same thing as finding a statistically significant one.
Q. But even a non-statistically significant increased risk is still an increased point estimate, correct?
A. An increased?
Q. Point estimate.
A. Well, the point estimate can be elevated. But it's not the same as saying that you've identified a risk.
Q. And even a non-statistically significant increased risk is still an increased risk?

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A. No. No, that's incorrect.
And I think you could wish for that to be true. But it would be chaos. It would be absolutely chaos if we just cherry-picked every single finding from every study that supported some idea that was pointing in one direction or another that didn't reach statistical significance. It would be chaos. How do you interpret that, then?
Q. A non-statistically significant increased risk would still be an increased risk that could be due to chance, correct?
A. Whether it's statistically significant or not, it can be due to chance, right; either one can be due to chance.
But this -- that would be one that failed the significance testing, which is part of doing epidemiologic studies.
Q. Doctor, you rely on non-statistically significant risk in

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your report, correct?
A. Did I rely or did I report on?
Q. You report on.
A. Well, sure, I tried to include what I find from the actual studies. Like, I'm trying to include the information and summarize it.
Q. So when you report that the Mansouri study found a protective effect for liver cancer in women, that was non-statistically significant, correct?
A. That's exactly right.
Q. And yet you still reported that finding; you didn't say there's no risk of liver cancer, you said protective risk -- or protective of liver cancer --
A. That's --
Q. -- correct?
A. That's the direction that it points. It was 0.9, which is less than 1. So it's -- it's even stronger evidence against there being a value that's above 1.

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But I wouldn't -- you know, if you ask the next question, about whether I would, then, interpret that as that women should start taking this to protect themselves against the risk of liver cancer, I wouldn't, and, in part, because that is not a statistically significant increase.

But that --

Q. So would --

A. I'm sorry.

Q. Sorry. I didn't mean to interrupt you.

A. I'm just saying that would only be one of many reasons that I wouldn't interpret it as truly finding that this -- that administration of that drug would be protective.

But -- but part of the information would be a non-statistically significant decrease below 1.

Q. When you use the terminology "protective," what would be your terminology on the other side, above 1?

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A. Elevated.

Q. Elevated. Okay.

So, Doctor, would you agree, then, that a -- for example, a 1.2 non-statistically significant result would be an elevated risk?

A. So the -- yeah. The estimate is elevated, that's right, above 1.

Q. And, Doctor, in the Mansouri study where you report the protective findings for women for liver cancer, they found an even higher elevated risk for liver cancer in men, correct?

A. Yeah. When you separate men from women, that's right. Because their initial analysis pooled the two, so it's a weighted average of both men and women together.

So for the women's value to go down, the men's value has to go up.

Q. Doctor, the Mansouri study doesn't report the increased risk of liver cancer in men when mixed users,

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those who use both exposed -- are both exposed to contaminated product and uncontaminated product, it doesn't report that -- that increased risk, correct -- or that risk?

A. I think so. But it's testing my memory. But I think what you're saying sounds right.

Q. Doctor, what are -- what are multiplicity analyses?

A. It's a little off from the term I would use.

But multiple comparison analysis, is that -- does that sound like what you're asking me?

Q. It does.

What's the terminology you would use since --

A. Yeah. I would take -- I'm sorry.

I would just say accounting for multiple comparisons.

Q. Okay. And what would the FDR test be in terms of comparing for

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multiple comparisons?

A. There's different techniques. And you're referring to a false discovery rate, right.

So it's -- but there's different techniques to be used in order to change the level of significance you're willing to accept by accounting for the number of different comparisons that you're making.

Q. There's different sections, one of those would be false discovery rate. Another would be Bonferroni, correct?

A. Bonferroni is an example. It accounts for a false discovery rate. I just --

Q. More --

A. I think the general --

Q. Go ahead.

A. I'm sorry. I apologize.

I just -- I just think the general concept is you're trying to reduce the risk of false discovery. And

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so Bonferroni is one technique that can be used.
Q. But there's also a technique called FDR, correct?
A. No, I don't think so. I mean, to me, that's -- that's the concept, right.
I think one of these studies used -- there was a technique by Hochberg and another colleague and -- which was another way to do the same thing. Like, there isn't just one way to do it.
But the concept is false discovery which means, you know, you've done so many tests that just by chance you're going to pop up positive ones that really aren't telling you the truth.
So you want to limit the chance of that happening by lowering the risk of false discovery.
Q. Doctor, the Bonferroni technique is rarely used in epidemiological studies assessing safety risk, correct?

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A. So I saw Dr. Sawyer say that. But I don't know that he's correct.
I mean, I've used it in studies. I don't know if it's fair to characterize it as rare. I mean, when I was initially, you know, being trained in biostatistics, it was really a very common one for people to describe.
And I have no idea what, you know, the prevalence of the use is. But it's still -- it's still out there to be used.
Q. Doctor, have you reviewed any papers that suggest the Bonferroni technique is inappropriate to use when assessing safety risk?
A. I don't know how it would pertain particularly to safety risk.
Q. And have you reviewed any papers that suggest that the Bonferroni technique is rarely used in epidemiological studies assessing safety risk in the past 20 years?

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A. I've not seen a paper that says that. I haven't sought one. The only -- the only information I have is from Dr. Sawyer saying that. And I don't know that he's correct.
Q. Doctor, have you analyzed whether or not the Bonferroni technique is rarely used in epidemiological studies assessing safety risk in the past 20 years?
A. Same kind of answer. I'm just -- I don't -- I don't know where I would go to find out what the prevalence is of the use of Bonferroni.
But I'm not aware that the qualifier "rare" would be true. I just don't know.
Q. Doctor, do you know whether or not other experts in this valsartan litigation have testified that the Bonferroni technique is rarely used in epidemiological studies assessing safety risk?
A. I don't know. I don't know

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the testimony of other -- other experts.
Q. Are you aware of whether or not other experts on behalf of defendants have testified that the Bonferroni technique is rarely used in epidemiological studies assessing safety risk?
A. So --
ATTORNEY DAVIDSON: Objection. Asked and answered.
THE WITNESS: Yeah. Sorry.
I still don't have awareness of what they've testified to.
BY ATTORNEY NIGH:
Q. Do you have any reason to disagree with them?
A. I mean, if -- I don't have any reason to disagree in the sense that if they can cite something, I would be happy to look at it. But it's not something I'm aware of I.
I mean, my colleagues still talk about using it, you know. So I don't know where the rareness idea comes

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from.
Q. When you say your colleagues still talk about using it, they talk about using it in assessing safety risk in epidemiological studies?
A. No. So here we're talking about, I guess, a pharmacoepidemiology study.
So I don't know about the state of, you know, what the preferred false discovery rate techniques are for safety studies.
But I'm talking about epidemiologic studies in general where the concept is generally the same, right. You do a lot of tests, you're going to get a lot of wrong answers. And you want to minimize the chance that that's happening.
Q. Bonferroni is assessing the chances of whether or not one or more of the results in a study may have been -- may have led to a false discovery or a false positive rate, correct?

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A. I don't accept that, no. I think -- I think it's a technique that's used to reduce the risk of that.
And so that what it does is it helps you to create a P value. So instead of the conventional .05, which is the 5 percent Type I error rate that you're baking into your study, it makes it something much smaller than .05. It's not just one specific number.
Q. So you think -- do you think it would be appropriate, in assessing safety risk, if there are 100 outcomes analyzed in an epidemiological study that you would divide the P value of .05 divided by 100?
A. I don't think that's the right technique. I don't think it's simple math like that.
There's -- there's other weighting that goes into it. And I don't conduct those analyses myself. So I can't describe to you in detail how the technique is actually applied.

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But I don't know. I mean, I don't know what opinion we're -- we're trying to augment here about something about safety.
I mean, I guess if the issue -- if the issue is about getting the truth, then you ought to take some care. If you don't care about the truth and you're willing to have a lot of false positives, and that somehow feeds into some idea about safety, I guess go for it, right.
But it's -- it doesn't -- it certainly doesn't enhance your ability to find the truth.
Q. So you don't believe that the way in which you find a Bonferroni score is to divide the P value divided by the number of outcomes assessed in the study?
A. I just don't -- I don't remember. I don't -- I don't conduct that part. Like, I work with biostatisticians in our studies. And so

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I don't -- that's not part of my work is to conduct it.
ATTORNEY DAVIDSON: A good time for a break?
ATTORNEY NIGH: I think so.
ATTORNEY DAVIDSON: You just seem to be --
ATTORNEY NIGH: No, now is no problem. I am moving to something different.
So let's take a ten-minute break.
VIDEO TECHNICIAN: Off the record, 1:43.
- - -
(Whereupon, a brief recess was taken.)
- - -
VIDEO TECHNICIAN: We are back on the record at 2:00 p.m.
ATTORNEY NIGH: Doctor, I do not have any other questions. Thank you.
THE WITNESS: Thank you.

ATTORNEY DAVIDSON: Sorry. I don't think I heard you.
ATTORNEY NIGH: I don't have any other questions.
ATTORNEY DAVIDSON: Okay. Let's just take a five-minute break. I think I might have one question, max.
VIDEO TECHNICIAN: Off the record, 2:00 p.m.
- - -
(Whereupon, a brief recess was taken.)
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VIDEO TECHNICIAN: We are back on the record at 2:02 p.m.
- - -
EXAMINATION
- - -
BY ATTORNEY DAVIDSON:
Q. Dr. Diette, you were asked a couple of questions earlier today about my client, ZHP.
Do you generally understand



what ZHP's role is in this lawsuit?
A. Well, yeah, I mean, I know they're -- they're a pharmaceutical company, obviously, from -- from China.
And I understand one of their products is valsartan that was contaminated with NDMA.
ATTORNEY DAVIDSON: I don't have any other questions.
ATTORNEY NIGH: Okay. No questions here. Thank you.
VIDEO TECHNICIAN: That concludes today's deposition. The time is 2:03 p.m.
- - -
(Whereupon, the deposition concluded at 2:03 p.m.)
- - -



CERTIFICATE

I, Amanda Maslynsky-Miller, Certified Realtime Reporter, do hereby certify that prior to the commencement of the examination, GREGORY DIETTE, Ph.D., was remotely sworn by me to testify to the truth, the whole truth and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing is a verbatim transcript of the testimony as taken stenographically by me at the time, place and on the date hereinbefore set forth, to the best of my ability.

I DO FURTHER CERTIFY that I am neither a relative nor employee nor attorney nor counsel of any of the parties to this action, and that I am neither a relative nor employee of such attorney or counsel, and that I am not financially interested in the action.

Amanda Miller
Certified Realtime Reporter
Dated: May 9, 2025

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You are signing same subject to the changes you have noted on the errata sheet, which will be attached to your deposition.
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ACKNOWLEDGMENT OF DEPONENT

I,_____, do hereby certify that I have read the foregoing pages, 1 - 155, and that the same is a correct transcription of the answers given by me to the questions therein propounded, except for the corrections or changes in form or substance, if any, noted in the attached Errata Sheet.

_____
GREGORY DIETTE, Ph.D. DATE

Subscribed and sworn to before me this _____ day of _____, 20____.

My commission expires:_____

_____
Notary Public

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LAWYER'S NOTES

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